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THE 1990 CANADIAN LONG-RANGE TRANSPORT OF AIR POLLUTANTS AND ACID DEPOSITION ASSESSMENT REPORT

Part 6

HUMAN HEALTH EFFECTS

1990

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FEDERAL/PROVINCIAL RESEARCH AND
MONITORING COORDINATING COMMITTEE (RMCC)

ASSESSMENT OF THE STATE OF KNOWLEDGE ON THE LONG-RANGE TRANSPORT OF AIR POLLUTANTS AND ACID DEPOSITION

PART 6: DIRECT AND INDIRECT HUMAN HEALTH EFFECTS

ENVIRONMENTAL HEALTH DIRECTORATE
HEALTH AND WELFARE CANADA

1990

TOXIC AIR POLLUTION - HEALTH EFFECTS SECTION
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6. Health Effects

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6.1 SUMMARY

6.1.1 POTENTIAL HEALTH EFFECTS FROM LRTAP

There is a broad range of health responses associated with exposure to air pollution ranging from the immediate such as aggravation of asthma and hospital admissions, to long term responses such as chronic lung disease, i.e. bronchitis. Subtle changes in respiratory health can be ascertained using indicators like decreased lung function, decreases in the rates at which inhaled particles are removed from the upper respiratory tract surfaces, and alterations in biochemical and immunological indices.

Studies (Schlesinger et al. 1983, Gearhart and Schlesinger, 1989) have reported changes in the connecting airways of rabbits which are thought to directly relate to the types of changes that are thought to cause bronchitis (Reid et al. 1983) after exposure to sulphuric acid mist at concentrations as low as 250 $\mu g/m^3$. Slowing of the clearance of particles out of the lung might well lead to increased infection, and longer times for particles to have adverse effects on airway tissue. Chronic exposure to ozone at levels of 0.25 ppm can produce structural changes in the lungs and can reduce total lung capacity in experimental animals.

Acute effects of sulphuric acid on humans are usually more pronounced in asthmatics, and occur rarely below 400 $\mu g/m^3$ (USEPA, 1988). Effects in normal adults have not been seen below about 900 $\mu g/m^3$ (Avol et al. 1988; USEPA, 1988). Asthmatics, however; are not any more sensitive to ozone than are non-asthmatics, although there is a wide range in sensitivity among healthy subjects (Linn et al. 1980; Solic et al. 1982; Linn et al. 1983; Shepard et al. 1983; Kagawa, 1984; Dreschler-Parks et al. 1987; Koenig et al. 1987; McDonnell et al. 1987; Reisenauer et al. 1988).

Sensitivity to ozone decreases following consecutive exposures (Hackney et al. 1977; Linn et al. 1980; Solic et al. 1982; Linn et al. 1983; Dreschler-Parks et al. 1987; Koenig et al. 1987; McDonnell et al. 1987; Reisenauer et al. 1988;), but returns after about a week of exposure to clean air (Horvath et al. 1981; Kulle et al. 1982). There is new evidence emerging that the effects of ozone appear to build up over a period of time much longer than one hour, i.e. a cumulative effect.

A recent study in Canada (Raizenne et al. 1989) observed small decrements in children's lung function which occurred during times of high air pollution. Acid levels reached a maximum of about 50 $\mu g/m^3$, and ozone levels were as high as 143 ppb. In two other Canadian studies (Stern et al, 1989; Raizenne et al. 1989) comparing respiratory health in a region of Canada which experiences high levels of LRTAP , with a region experiencing very low levels of LRTAP, an average 2% decrement in children's lung function was observed in the more polluted region. Children in the more polluted area also had a higher incidence of upper respiratory infections. It is believed that the observed difference is likely due to the coexistence of acidic aerosols and other

pollutants, i.e. O_3, SO_2.

Hospital admissions for respiratory conditions are indicative of trends in respiratory health in the general population. Bates and Sizto (1983, 1986, 1987) have found an association between admissions in summer for respiratory disease and daily pollution levels. In analyzing their data they propose that sulphuric acid aerosol is the pollutant most probably responsible for this association. They also propose that the combination of ozone and acid may increase individual susceptibility.

Laboratory studies of interactions between acid aerosols and ozone have not provided overwhelming evidence of possible synergism. In human clinical trials using mixtures of sulphuric acid or sulphur dioxide and ozone, the observed effects have generally been attributed to ozone (Bates and Hazucha, 1973; Hazucha and Bates, 1975; Bell et al. 1977; Bedi et al. 1979; Kleinman et al. 1981; Bedi et al. 1982; Kulle et al. 1982; Stacy et al. 1983; Folinsbee et al. 1985; Kagawa, 1986; Horvath et al. 1987). In animal studies, synergistic or non-synergistic interactions have been observed between acid aerosols and ozone dependent upon the endpoints used for the studies. Using morphology as an indicator of interaction, synergism has not been observed, the effects were attributed to ozone (Cavender et al., 1977, 1978; Juhos et al., 1978; Moore and Schwartz, 1981). However, when biochemical and mucus secretion were used as endpoints, synergism was observed (Last and Cross, 1978; Last et al. 1983; 1984; 1986; Warren and Last, 1987). While direct extrapolation from experimental animals to humans is not possible, the animal studies do indicate that synergism may be occurring at a more subtle level than observed in the human studies.

6.1.2 REGIONAL VARIATION OF EXPOSURE IN CANADA

A rough measure of the amount of pollutant to which Canadians are exposed out of doors can be obtained through monitoring of air quality. Very little monitoring has been done for acid levels in Canada, however there is an extensive data base on ozone.

In Canada, the highest acid measurements were taken in southwestern Ontario and Nova Scotia. Monitoring in Penticton, British Columbia, showed very low levels of acid. The highest measurements to date were observed during a summer camp study on the north shore of Lake Erie, reaching a maximum hourly sulphuric acid level of approximately 50 $\mu g/m^3$ (Keeler et al. 1989).

The Canadian 1-hour maximum acceptable ozone level of 82 ppb is often exceeded along the Windsor-Quebec City corridor, and occasionally in the Vancouver area. Levels observed in the United States are often considerably higher.

6.1.3 HEALTH CONCERNS OF LRTAP

The concern warranted by current levels of LRTAP in Canada depends to a large extent on the mode of action these pollutants exert on the respiratory system. Hattis (1990) postulated that acid particles act by stimulating localized irritation of the lining of the connecting tubes. Therefore it is plausible that very small effects, like those observed in animal studies, could continue to accumulate over a long period of time. This has implications for the interpretation of epidemiological studies which have observed small decrements in children's lung function. It could imply that the lung functions of the children will continue to decline or decrease their growth rates with continuing exposure to acid aerosols.

Ozone already occurs in some parts of Canada at levels which are known to cause transient respiratory effects in healthy people. The effect of combinations of pollutants may also be of concern. It has been suggested (Bates and Sizto, 1989) that ozone may increase susceptibility to other pollutants.

6.1.4 INDIRECT HEALTH EFFECTS FROM ACID DEPOSITION

Human health may be affected by acid deposition in an indirect manner. The acidification of water supplies and soil with subsequent mobilization of heavy metals may increase exposure to these metals through drinking water and food. Of significant interest are cadmium, mercury, lead, arsenic, aluminum and chromium. Cadmium and mercury may enter into food sources and may, as in the case of mercury, result in biomagnification through the food chain to man. Very low doses of lead can have subtle effects on the central nervous system that are observed as peripheral nerve dysfunction in adults or neurobehavioural and developmental effects in children.

Unregulated water supplies are not pH neutralized before distribution and increased acidification of the water can result in corrosion of the distribution system. The corrosion of metal plumbing systems can result in increased levels of potentially toxic heavy metals in the water. In Ontario, elevated levels of dissolved copper, lead, and cadmium have been observed in water that was left undisturbed in distribution systems drawing water from privately supplied lake water (Health and Welfare Canada, 1987; Meranger et al. 1983). Flushing of the systems for five minutes reduced the concentrations of these metals. In areas sensitive to acid deposition, the corrosive effects can be severe in untreated water systems. Shallow wells may be especially susceptible to increases in metal concentrations and drilled bedrock wells may not be immune as heavy metals, like arsenic, may be mobilized from mineral sources into aquifers (Meranger et al. 1983; Cherry, 1984). Northern Ontario, southwestern Ontario, the north shore of the St. Lawrence River in Quebec and areas of New Brunswick and Nova Scotia are areas in Canada that are very sensitive to acidification.

Currently, the most important effect that acidification of water supplies may present to human health is from the release of potentially toxic heavy metals from metal plumbing systems. This would especially be the case in unregulated water distribution systems in acid sensitive areas of Canada. Due to its subtle toxic effects and its use in plumbing systems, lead may represent the greatest threat to human health from increased acidification of untreated water supplies. Increased levels of methylmercury in water may occur from acidification and subsequently contaminate food. The toxic effects of methylmercury, however, can be avoided by reducing or eliminating consumption of excessively contaminated food sources.

6.2 DIRECT HEALTH EFFECTS OF LRTAP

There is increasing evidence that chronic exposure to acid particles and other transported air pollutants is associated with modest but measurable effects on human health. This report examines current research findings and attempts to provide a risk perspective of acid air pollution in Canada. The report focuses on acid aerosols and ozone, the two components of LRTAP most likely to be affecting human health. In trying to answer the question: What are the potential health effects which may be caused by LRTAP?; the review includes data from animal toxicology, human clinical studies, and epidemiological studies. Some theories are also proposed for how these pollutants may act on the respiratory system.

The second part of the report examines monitoring data for acid aerosols and ozone in different regions of the country. The third section will assess the exposure information and the health effects information to provide a statement on the risk posed to the health of Canadians by LRTAP. The final part identifies ongoing research needs and summarizes current Canadian research projects.

6.2.1 POTENTIAL HEALTH EFFECTS OF LRTAP

6.2.1.1 RANGE OF HEALTH RESPONSES TO AIR POLLUTION

Direct health responses to the inhalation of airborne pollutants can be acute (short-term) or chronic (long-term). Acute responses are those occurring immediately or within a few days of exposure. Chronic responses may take months or years of exposure before becoming noticeable. Chronic responses usually appear as increased rates of health deterioration or increased incidence of disease in exposed populations. There is also a wide range in the severity of responses. Respiratory illness spans a variety of diseases, the most serious of which are emphysema, chronic bronchitis and asthma--collectively referred to as chronic obstructive lung disease. Other clinical symptoms indicative of upper or lower respiratory tract infections include chronic coughing, cough with phlegm, croup, substernal chest pains, pneumonia, chest colds, wheezing, and allergic responses

to inhalant particles such as dust, animal fur or mould. Diminished lung function is a more subtle indicator of respiratory morbidity. Pathological changes in lung tissue such as decreases in mucociliary clearance rates (the rate at which inhaled particles are removed from upper respiratory tract surfaces) and alterations in biochemical and immunological indices, are also indicative of respiratory morbidity. Other effects of air pollution may include the aggravation of existing disease, such as asthma; increased severity of respiratory infections; and accelerated ageing.

6.2.1.2 RECENT STUDIES: ACID AEROSOLS

Schlesinger et al.(1983) have reported increased concentrations of mucus secreting cells in connecting airways of rabbits two weeks after the end of a four week exposure to submicrometer sulphuric acid mist at concentrations ranging from 250 to 500 $\mu g/m^3$. This finding is significant because it relates directly to the types of changes that are thought to be associated with chronic bronchitis (Reid et al. 1983). In these experiments there were also changes in the speed with which foreign matter was cleared from the rabbits' connecting airways. The effectiveness of particles in causing these changes in clearance appears to depend directly on the acidity of the particles--acid sulphate salts and sulphuric acid cause the effect whereas neutral sulphate salts do not (Schlesinger, 1984).

Most recently, Gearhart and Schlesinger (1988) have confirmed the initial finding of elevations in mucus secreting cells in response to 1 hour/day sulphuric acid exposures of 250 $\mu g/m^3$, and shown that some of this change persists 3 full months following the end of a 1 year exposure. They also observed effects on clearance function, and it appeared that this change in function of an important lung defense system showed effects that were more pronounced 3 months after the end of exposure than immediately after the end of exposure. Such slowing of the transport of foreign particles out of the lung might well lead to increased infection, and to longer residence times for particles (including non-acid types of particles) to produce adverse effects.

Although, acute effects on lung function in adolescent asthmatics have recently been reported at concentration of sulphuric acid as low as 68 $\mu g/m^3$ (Koenig et al. 1983) most results in asthmatics have been negative below 400 $\mu g/m^3$ (USEPA 1988). Pulmonary function responses have not been observed in normal adults below about 900 $\mu g/m^3$ (USEPA 1988; Avol et al. 1988).

A recent Canadian study (Raizenne et al. 1989) examined the lung function of girls attending summer camp in a region of Ontario which periodically receives high levels of acid pollution. There were four distinct periods of high acid aerosol, maximum levels observed were 47.7 $\mu g/m^3$ sulphuric acid. Maximum levels of ozone were recorded at 143 ppb. During one particular episode small decrements in lung function were observed.

Stern et al.(1989) have compared the lung functions of 7-12 year old children in two Canadian communities--Tillsonburg, Ontario, located in a region with elevated concentration of transported air pollutants, and Portage la Prairie, Manitoba, located in a relatively low pollution area. They found that Tillsonburg children had statistically significant lower levels of Forced Vital Capacity (FVC) and Forced Expiratory Volume ($FEV_{1.0}$)($p < .001$) than did those in Portage la Prairie. These differences could not be explained by parental smoking or education, cooking or heating fuels, pollution levels on day of testing or differences in age, sex, height or weight. The differences persisted when children with either cough with phlegm, asthma, wheeze, inhalant allergies or hospitalization before age 2 for a chest illness were excluded from analysis. With the exception of inhalant allergies, which occurred more frequently in Tillsonburg children, the prevalence of chronic respiratory symptoms and illness was similar in the two communities.

It is not possible to attribute the apparent change in lung function to any specific pollutant or combination of pollutants, however it is worth noting that the two communities differed far more in their sulphate exposures (a rough proxy for acid) than they did in other measures of pollution (see Table 2 in Stern et al. 1989). In particular, the two areas showed only a very modest difference in long-term average ozone levels, although the Tillsonburg area has many more days on which peak ozone levels exceed the 1 hour standard of 0.08ppm. The study was done during the winter months when both acid and ozone exposures are low--lessening the chance that the observed differences reflect short-term responses. Follow-up studies in 5 communities in Saskatchewan (Stern et al. submitted), a low LRTAP area, and 5 communities in southwestern Ontario, a region experienceing relatively high levels of LRTAP, confirmed these results. On average a 2% decrement in lung function was observed in the Ontario children. The larger sample size and study of several communities within the areas lends greater support to the earlier findings.

In a study of hospital admissions in Southern Ontario, Bates and Sizto (1983; 1986; 1987) found an association between admissions in summer for respiratory disease and daily levels of sulphate, ozone and temperature. No association existed for non-respiratory admissions. Analysis of individual pollutant data indicated that sulphate had the highest correlation with admissions, yet sulphate is not considered to be an irritant at the levels measured. Levels of ozone, which have been shown to irritate airways occurred, however; there was no unusually high admissions associated with days of high ozone levels. Bates and Sizto suggest (1989) that some other pollutant, most likely sulphuric acid aerosol is responsible for the observed association, and that the combination of ozone and acid can increase individual susceptibility.

6.2.1.3 RECENT STUDIES: OZONE

The primary oxidative damage caused by ozone leads to leakage of fluid, containing blood proteins into the alveolar spaces. This has been detected in both animals (Bassett

et al. 1988) and humans (Kehrl et al. 1987). Fluid also builds up underneath the damaged membranes (Yokoyama et al. 1989). Either this, or the release of chemical mediators causing contraction of the muscles surrounding the airways, may be the cause of the relatively short-term changes in measures of pulmonary function (FVC and $FEV_{1.0}$) that have been observed.

This oxidative damage also leads to the migration into the alveolar spaces of macrophages and neutrophils. In a manner similar to the defensive response to cigarette smoke, the enzymes released by these cells can also digest healthy lung tissue, doing some additional damage to the lining of the alveoli and bronchioles, and to elastin and collagen, very large proteins within the lung tissue that help to provide important support to the whole structure. Through this sequence of events it is plausible that ozone concentrations that induce acute inflammatory response could contribute to the long-term development of emphysema and related chronic lung diseases.

Recent studies in both rats (Grose et al. 1989) and monkeys (Hyde et al. 1989) indicate that chronic exposure to moderate levels of ozone (about 0.25 ppm, given in various schedules) can produce structural changes in the lung and can reduce total lung capacity.

In recent comprehensive reviews, Lippman (1989a and 1989b) has made the following general points on the reversible effects of ozone:

* Ozone related decreases in lung function increase with increasing exercise (Hazucha, 1987).

* There are wide differences in sensitivity among healthy subjects, although sensitivity is not associated with other lung risks or adverse conditions, such as smoking, old age, asthma, upper respiratory allergies, and chronic obstructive lung disease (Linn et al. 1980; Solic et al. 1982; Linn et al. 1983; Shephard et al. 1983; Kagawa, 1984; Dreschler-Parks et al. 1987; Koenig et al. 1987; McDonnell et al. 1987; Reisenauer et al. 1988).

* Repeated exposures on consecutive days produce successively weaker responses, until by day 5 there is essentially no response (Hackney et al. 1977; Linn et al. 1980; Solic et al. 1982; Linn et al. 1983; Dreschler-Parks et al. 1987; Koenig et al. 1987; McDonnell et al. 1987; Reisenauer et al. 1988). Responsiveness returns after about a week of exposure to relatively clean air (Horvath et al. 1981; Kulle et al. 1982).

* The biological effects of ozone appear to build up over a much longer period of time than 1 hour (Folinsbee et al. 1988).

* The duration of exposure has an effect on the magnitude of response. There is an indication that duration of exposure is synergistic with concentration in situations where ozone levels are low and duration of exposure is prolonged.

* The buildup of ozone related effects over extended daily exposures apparently results in greater lung function decrements in field studies than in subjects exposed to comparable concentration in chambers for 1 or 2 hours (Spektor et al. 1988; Kinney et al. 1988; 1989).

* Children and adults appear likely to receive roughly similar ozone doses to various regions of the lung, given similar activity levels (Miller and Overton, 1989) and given similar activity, similar declines in FVC and $FEV_{1.0}$ have been observed (McDonnell et al. 1986).

Long-term epidemiological studies in two communities in the Los Angeles, California area have provided suggestive evidence that the rate of loss of lung function as individuals age is greater in the community with the higher levels of ozone (Detels, 1987). Lippmann (1989b) has recently gone farther with these data, juxtaposing the results of the two Los Angeles area communities with analogous observations by a different set of investigators for the relatively clean-air community of Tucson, Arizona (Table 6.2.1). He concluded that the communities with higher air pollution levels were at much greater health risk than relatively clean communities. Nevertheless, these suggestive differences among communities cannot be definitively attributed to ozone alone.

6.2.1.4 MECHANISMS OF ACTION: ACID AEROSOL

Acid particles enter the respiratory system as a combination of solid material and liquid (depending on the outside temperature and humidity). Additional water quickly condenses on the incoming particles from the moist air in the upper respiratory system. The increased size of the particles due to this condensation tends to increase the deposition of the particles in the connecting airways of the lung--from the trachea through the smaller branching tubes leading to the bronchioles and the alveoli. The surface of these connecting airways normally is somewhat protected by a layer of mucus, although in the smallest connecting airways deep in the lung it is likely that the mucus is not continuous, but in the form of small isolated droplets (Van As, 1977). The mucus and the underlying cells have a certain capacity to neutralize acid delivered by incoming particles (Holma, 1985; 1989), but localized adverse effects may occur when, despite this neutralizing capacity, the local acidity is pushed to levels that cause irritation or damage (Holma et al. 1977).

Theoretical calculations (Hattis et al. 1987; Holma, 1989) make it appear unlikely that overall acidification of the mucous lining could occur at airborne sulphuric acid levels much lower than a few hundred micrograms per cubic meter--well above ambient

Table 6.2.1

Annual Change in Lung Function

Population (number)	$FEV_{1.0}$ (ml)	FVC (ml)	$FEF_{25-75\%}$ (ml/sec)	V_{50} (ml/sec)	V_{75} (ml/sec)
Males					
Tucson, Az. (86)	-29	-30	-36	-37	-23
Lancaster, Ca.(153)	-46	-51	-47	-65	-44
Glendora, Ca. (168)	-48	-60	-89	-112	-69
Females					
Tucson, Az. (176)	-19	-17	-31	-24	-25
Lancaster, Ca.(286)	-33	-38	-53	-77	-41
Glendora, Ca. (325)	-44	-44	-97	-109	-76

$FEV_{1.0}$ - forced expiratory volume in one second
FVC - forced vital capacity
$FEF_{25-75\%}$ - flow rate between 75 and 25% of vital capacity during FVC
V - mean minute ventilation

from Lippmann (1989b)

concentrations. Observations of short-term changes in lung function in experimental clinical studies generally seem to require quite high levels of exposure. Because of this, Hattis et al. (1987; 1989) have proposed that the most likely mechanisms for effects at environmentally-relevant dose rates would have to involve a chronic accumulation of irreversible or slowly reversible effects resulting from localized responses of the surface of the connecting airways to individual acid particles. The rate of accumulation of the effects from these localized responses is anticipated to depend on the number of these particles which are large enough to:

* survive passage through the upper respiratory system without being neutralized by ammonia (which is sometimes present in relatively high concentration in the mouth);

* be deposited on the surface of the connecting airways; and

* create a large enough change in local pH to be detected by the underlying cells.

If the only viable hypothesis was that acid-produced effects occur when the overall mucus in the connecting airways of the lung is acidified, then the acid effects would necessarily have a sharp threshold, and only be likely at relatively high concentrations. Because acid/base reactions are very fast, and the lining of the lung is continuously resupplied with additional acid-neutralizing capacity from the blood stream, acid exposures would need to exceed levels over a very brief time (minutes or at most an hour) or not have an effect at all. If on the other hand, acid-bearing particles can have effects by delivering localized "irritant signals" to small areas of the lining of the connecting airways, then it becomes possible for the effects of acid particles individually to contribute to small effects regardless of the overall concentration of acid particles present. Moreover, the localized effects could accumulate over a considerable time, depending on the period required for the localized biological responses to reverse themselves. For a response like the proliferation of mucous-secreting cells the effective period for reversal might well be many months or years in humans.

6.2.1.5 MECHANISMS OF ACTION: OZONE

Ozone is a reactive gas that is poorly soluble in water. Because of its low solubility, most of the ozone that is breathed in evades the body's first line of defense (the moist passages of the nose and other respiratory airways) and penetrates the respiratory system (Hatch et al. 1989). The primary damaging reactions occur on and beneath the lining of the cell membranes, along the respiratory passages and into the alveoli.

Fortunately, there are some chemical defenses against ozone's tendency to oxidize fatty acids. Systems that incorporate vitamin E and glutathione, in particular, tend to either prevent or reverse the primary oxidative damage caused by ozone, and terminate the destructive free radical chain reactions that ozone initiates (Menzel and Wolpert, 1989). These defensive systems, however; cannot operate so efficiently as to prevent or immediately reverse all damage, even at very low exposure levels. Some of ozone's free radical progeny will encounter fatty acid molecules before they encounter detoxifying defensive molecules. Once the oxidized fatty acids are generated within the membranes, they will persist for some time until repair systems find them. Thus there are likely to be some changes at the molecular level due to ozone exposure, even at the lowest conceivable ozone concentrations. At higher ozone exposure level, however; as the unused portions of the body's defensive systems become depleted, relatively more damage is done per unit of ozone exposure and, other things being equal, damage may persist for a longer time. Thus as ozone concentrations are increased to relatively high levels, the amount of damage may increase more than proportionately.

6.2.1.6 INTERACTIONS BETWEEN OZONE AND ACID AEROSOLS

The atmosphere that humans are exposed to in day to day life is a complex mix of compounds including man-made acid aerosols and ozone. Thus, while studies of exposures to single compounds are useful as a method to observe effects specifically due to a particular compound they do not represent "real life" situations. Studies of mixtures of compounds do not represent real life either but they can indicate whether compounds may synergistically interact to cause more deleterious effects than may be caused by exposure to single compounds.

In animal studies, the combination of acid aerosols, usually sulphuric acid (H_2SO_4), and ozone (O_3) have given indications of synergism and non-synergism dependent upon the endpoint of the study. Using morphological endpoints, interaction did not appear to occur between the pollutants; the effects observed were due to the ozone (Cavender et al., 1977, 1978; Juhos et al., 1978; Moore and Schwartz, 1981). However, using biochemical endpoints or increased mucus secretion, researchers have observed synergism between acid aerosols and ozone (Last and Cross, 1978; Last et al. 1983; 1984; 1986; Warren and Last, 1987). Grose et al. (1982) observed an increase in bacterial infectivity when mice were exposed to ozone and sulphuric acid compared to either compound alone. The animals in the simple mixture studies were exposed to concentrations of sulphuric acid that ranged from 40 to 1000 $\mu g/m^3$ and ozone concentrations from 0.1 to 2 ppm. Complex mixtures that contain acid aerosol species and ozone indicate some level of interaction. Mautz et al. (1985) observed a greater change in resistance and compliance in dogs exposed to atmospheres containing sulphuric acid plus ozone than in atmospheres containing ozone. These complex atmospheres also contained iron and manganese sulphates and sulphur dioxide (SO_2). It should be noted that there is a possibility that a mixture of SO_2 and O_3 may result in the formation of H_2SO_4.

The effect of mixtures of acid aerosols (H_2SO_4) and ozone in human clinical studies does not indicate interaction between the two compounds. The pulmonary function responses were attributed to ozone and were not altered by sulphuric acid (Kleinman et al. 1981; Kulle et al. 1982; Stacy et al. 1983; Kagawa, 1986; Horvath et al. 1987). These observations were made regardless of whether the exposures were simultaneous or sequential. The effects of the combination of sulphur dioxide and ozone (possibly forming sulphuric acid) has been investigated in human clinical studies (Bates and Hazucha, 1973; Hazucha and Bates, 1975; Bell et al. 1977; Bedi et al. 1979; Kleinman et al. 1981; Bedi et al. 1982; Folinsbee et al. 1985). Except for the study by Hazucha and Bates (1975) that suggested possible interaction, no synergism from the exposures was observed. The responses observed were attributed mainly to ozone.

In a similar light, the combination of ozone and nitrogen dioxide may, under the proper conditions, lead to the formation of nitric acid. Nitric acid may be important in considering acid aerosol-related health effects due to its acidity (Fine et al. 1987). Koenig et al. (1988)

suggested that acute exposure to nitric acid may cause a decrease in the pulmonary function of adolescent asthmatics. However, in controlled experimental conditions, NO_2-O_3 mixtures have had either synergistic effects or non-synergistic effects in animals depending upon the endpoints being measured and the exposure protocol (Freeman et al. 1974; Ehrlich et al. 1977; Watanabe et al. 1980; Veninga et al. 1982; Mustafa et al. 1984). Using bacterial infectivity in mice as an endpoint, Ehrlich et al. (1977) observed synergistic effects from the mixture whereas the individual components had no effect. In similar studies, Gardner et al. (1982) and Graham et al. (1987) used bacterial infectivity in mice to observe synergism between NO_2 and O_3 when the initial atmospheres were spiked with extra NO_2 and O_3. Synergistic effects were observed when the spike concentrations exceeded 1880 $\mu g/m^3$ NO_2 and 196 $\mu g/m^3$ O_3. Freeman et al. (1974) observed lesions in the lungs of rats that were attributable primarily to the ozone and not to interaction between NO_2 and O_3. In human clinical studies, observed effects from exposures to NO_2 and O_3 were attributable to the ozone and interaction was not observed (Hackney et al. 1975a; 1975b; Folinsbee et al. 1981; Kagawa, 1983; Adams et al. 1987). The effects observed in the human clinical studies were generally additive, that is, the effects created by the inhalation of the compounds was not greater than would be expected from simply adding together the effects from exposure to the individual compounds.

Most of the human clinical studies have used lung function tests to determine the effects of combined exposure to the pollutants in question. Whether lung function testing is a sensitive indicator of adverse health effects is unsure. These testing protocols reveal changes in airway resistance and lung volume while more subtle adverse effects may be occurring at a cellular level. While animal studies can use more invasive techniques to study the effects of interaction it is often difficult to extrapolate these effects to the human situation.

6.2.2 REGIONAL VARIATION OF EXPOSURES IN CANADA

6.2.2.1 ACID AEROSOLS

The extent of our present knowledge on the magnitude, frequency, and duration of acidic pollutant episodes is essentially non-existent for most regions of Canada. Since most of the public focus and scientific study on the topic of acid deposition and long range transport has centred in eastern Canada, the majority of our knowledge on the levels of ambient acids is derived from indirect acidic aerosol measurements taken in this region. While measurements of the basic "criteria pollutants" such as sulphur dioxide, ozone, etc., have been performed for more than 15 years in Canada for the National Air Pollution Surveillance (NAPS) network, the measurement of aerosol acidity has only recently been performed in a few locations in Canada by National Health and Welfare (Smith-Palmer and Wentzell, 1986; Keeler et al., 1989;1990). Measurements of nitric acid, on the other hand, have been made in selected locations across Canada over the past decade as reliable

measurement techniques were developed (Barrie and Sirois 1980; Anlauf et al., 1985). While the lack of specific aerosol acidity data in all areas of Canada limits the present discussion somewhat, available emissions and ambient measurement data can be used to explain the physical and chemical processes that generate the acid exposures. This information in turn allows one to draw some limited inferences about likely exposures in those areas of Canada in which only emissions, meteorological, and criteria pollutant data are available.

We will first examine available aerosol acidity data and discuss the patterns of exposure in those areas of Canada. These exposure patterns will then be compared to those observed in neighbouring regions of the United States.

Human exposure to acidic aerosols simply expressed as the concentration (C) times time (T), (C x T), may not be ideal for representing realistic patterns of exposure, or for predicting biological responses. The aerosol acidity concentration that a region will experience at any one time is a complex function of the upwind pollutant emissions, distance from the sources and meteorological history of the air masses reaching the region. The aerosol acidity may also be a function of the available ambient ammonia that interacts with the acidic air mass.

In practice, the aerosol acidity measured in the atmosphere in specific places will be the net result after the combined influence of all of these processes. Human exposure will then be the interaction of this acidity with variations in lifestyle and differences in the concentrations observed in the various "microenvironments" where a person spends time. While this resulting "personal exposure" to pollutants is a critical concern, it is beyond the scope of the present discussion and is the subject of extensive scientific assessment. Since the indoor acid aerosol concentrations can be assumed to originate from the outdoors in most of Canada and the United States, we will limit our discussion to measured outdoor concentrations.

In general, the highest aerosol acidity concentrations measured to date have been in the regions that have the highest density of sulphur dioxide emissions--the midwestern United States and southeastern Canada. This region records the highest ambient sulphate concentrations as well as the most acidic rainfall (Clark, 1980). While the observation of very low aerosol sulphate concentrations indicates low aerosol acidity, elevated sulphate in a region only indicates that it may potentially, but not necessarily, be an area of elevated aerosol acidity; it does not guarantee it to be true. Aerosol sulphates commonly exist as completely neutralized ammonium sulphate. Partially neutralized acidic sulphates, such as ammonium bisulphate and sulphuric acid, are the primary species of concern at the present time.

6.2.2.1.1 Aerosol Acidity Measurements In Ontario

The first measurements of aerosol acidity in Ontario were made during the Canadian Acute Respiratory Effects Study, 1986 (CARES '86) (Raizenne et al., 1989; Spengler et al., 1989). This field health study assessed the acute health effects of air pollutants on children at a girls summer camp on the northern shore of Lake Erie in SW Ontario. Measurements of various pollutants including ozone and aerosol strong acidity were taken for the 42-day period from 29 June through 9 August, 1986. The measured aerosol was periodically quite acidic with observed 12-hour averaged H^+ (hydrogen ion) concentrations reaching as high as 560 nmoles/m^3. Acid events occurred once every two weeks lasting for 2-3 days in duration. The aerosol acidity concentrations measured during the study have been reported by Keeler et al. (1990). The acid event which was observed on 25 July recorded H^+ concentrations above 100 nmoles/m^3 (equivalent to about 5 $\mu g/m^3$ of sulphuric acid) for 4 consecutive 12-hour samples and the continuous sulphate instrument recorded an hourly sulphuric acid concentration maximum of 50 $\mu g/m^3$ (Keeler et al., 1989).

A year long aerosol acidity record has now been obtained at two sites in Canada, Dunnville and Pembroke, Ontario, as part of a joint project between the Harvard School of Public Health and researchers at National Health and Welfare Canada (Keeler et al., 1989). The aerosol acidity measured in Dunnville is higher than that seen in Pembroke, Ontario. At both sites, approximately 50% of the sampling days in a one year period were found to have no detectable acidity (H^+). However, the Dunnville site observed H^+ concentrations greater than 100 nmoles/m^3 (equivalent to 5 $\mu g/m^3$ H_2SO_4) on 8% of the total sampling days, whereas the Pembroke site observed this amount on only 1% of the days. This can be contrasted to a Hendersonville, Tennessee, site where no detectable acidity was observed for only 22% of the sampling days, and H^+ concentrations greater than 100 nmoles/m^3 were observed for over 12% of the days.

During the period from 23 July to September 1, 1988, samples were also being taken in Egbert, Ontario, which is northwest of Toronto (Keeler et al., 1989). Aerosol acidity values measured at this site were generally lower than were found in Dunnville but higher than were found in Pembroke.

Waldman et al. (1989) measured the aerosol acidity in Toronto during the summer of 1986. Dichotomous samplers were utilized with ammonia denuders placed at the entrance of the samplers. Samples were collected for six weeks from 2 July through 10 August, 1986. Samples were taken twice daily from 0900-1700 and from 1700-0900 providing an 8- and 16-hour sampling interval. The observed aerosol was found to be periodically acidic and the sulphate aerosol was found to be 10 to 40% acidic. Maximum concentrations of aerosol acidity were 170 to 400 nmol/m^3, more acidic than those taken in Toronto. It is apparent there can be large differences in aerosol acidity across relatively small distances. The position of the Great Lakes along the U.S.-Canadian border is a major reason for the spatial differences observed in this region.

It is thought that acidic aerosol levels are lower in urban areas because of higher urban levels of ammonia. In Waldman's study (1989), they observed that on days when acidity was low, the downtown site had the lowest acidity, but that during higher acid episodes acid levels at the various sites were quite similar. It appears that for the higher acid episodes, the higher levels of ammonia that are thought to occur in urban areas may not be sufficient to neutralize a significant amount of acidity in cities the size of Toronto.

6.2.2.1.2 Acidic Aerosols In Other Provinces

Smith-Palmer and Wentzell (1986) measured the ambient aerosol acidity in Nova Scotia during two periods; 18 August through 11 September, 1983, and 23 May through 9 July, 1984. 24-hour samples were collected using dichotomous samplers with no ambient ammonia removal upstream, which may have caused an underestimate of the true acidity. The mean and standard deviation of the measured acidity, expressed as $\mu g/m^3$ sulphuric acid equivalent, were 0.6, 1.7 and 0.5, 1. The maximum 24-hour acid value recorded in the study was approximately 190 $nmol/m^3$, giving a ratio of hydrogen ion to sulphate of 0.33. This indicates that while the aerosol was acidic, a large portion of the sulphate was in a neutralized state. This may also indicate that some neutralization occurred on the filter media after collection. While these data suggest that the aerosol was acidic in Nova Scotia at the time of measurement, the sampling was not performed throughout the months one would expect the highest acidities.

Measurements of aerosol acidity have not been performed in many areas of Canada. Currently, acidity observations that are being made in Penticton, B.C. are showing very little acidity, with no peaks above 40 $nmol/m^3$ to date (Keeler et al., 1989). While there may be isolated, non-point source influenced locations outside of Ontario where significant aerosol acidity would be observed, the highest outdoor exposures to aerosol acidity are probably found in Ontario and possibly Quebec.

6.2.2.1.3 Day-Night Variations In Aerosol Acidity

The temporal patterns of aerosol acidity are not well understood. Limited measurements of day vs nighttime levels have not provided a clear picture of what to expect. At high altitude there appears to be no diurnal variations in either the H^+ concentration or the H^+/sulphate ratio (Pierson et al., 1989). At other surface or low altitude sites there is not a consistent pattern. For example, Keeler et al. (1989) observed a day/night pattern in Zanesville, Ohio during the summer of 1988 from the measured aerosol H^+/sulphate mole ratio. From their data, the aerosol appears to be consistently more acidic during the daytime than at night. However, this pattern is not as clear at Egbert, Ontario where the H^+ concentration and the H^+/sulphate mole ratio is often just as high at night as during the preceding day (Keeler et al. 1989). The lack of a clear day/night difference in these data may be explained by the large distance from the sources to Egbert. Transport of acidic air masses would experience at least one diurnal cycle before reaching the site, whereas the Zanesville site could be affected by air masses that were less than one day

old.

6.2.2.1.4 Several-Hours/Days "Episodes" Of Relatively High Acid Aerosol Exposure

Acid aerosol events have been observed at several locations in Ontario and Nova Scotia. As noted earlier, an exposure dose can be simply written as a function of concentration and time (C x T). As a first approximation, we will simply utilize the CxT product to give us an overall summary of exposure. As discussed earlier, chronic bronchitic-like effects have been observed in rabbits exposed repeatedly 1 hour/day to sulphuric acid aerosol at 250 $\mu g/m^3$ (Schlesinger et al., 1983; Gearhart and Schlesinger, 1988). Although there are no observations of concentrations this high at any one time, if the effects accumulate over periods of a day or more, then it may be relevant that sulphuric acid exposures greater than 250 ($\mu g/m^3$)-h/day may occur in the ambient environment (Table 6.2.2). Raizenne and Spengler (1989) have used the C x T formula to construct individual exposure estimates of children using the pollution data from CARES '86. Although no significant relationships between estimated acid dose and lung function were reported, the authors noted that the variability in exposures (from <500 $\mu g/m^3$ to >1500 $\mu g/m^3$) was very large in a group of children having essentially the same activity pattern.

In conclusion, exposures to acidic aerosols in eastern Canada appear to be somewhat lower than those in parts of the midwestern United States, based upon recent measurements. However, maximum exposures in southern Ontario are greater than 250 $\mu g/m^3$-h/day--equivalent, on a daily basis, to levels that have produced chronic bronchitis-like changes in rabbits (Schlesinger et al., 1983; Gearhart and Schlesinger, 1988). The regions of Canada with the highest sulphur dioxide emissions densities should be further investigated for elevated aerosol acidity. This is especially true for southern Ontario and southern Quebec, where the population density is greatest and the potential for LRTAP is also greatest.

6.2.2.2 OZONE

Unlike the situation that now exists for acidic species in the atmosphere, the data base that has been gathered for ozone is fairly comprehensive. Within the northeastern United States and Canada, the highest ground-level ozone concentrations attributable to human activity occur during the summer months. Isolated occurrences of extreme ozone levels are occasionally observed in other seasons, particularly the winter and spring, but these are more likely to be the result of intrusion of stratospheric ozone rather than to photochemical reactions among the chemicals released by automobiles and industry.

Environment Canada reports have described the relative frequency of elevated ozone concentrations in different regions of Canada (Dann, 1989). The Canadian 1-hour maximum acceptable level is often exceeded along the Windsor-Quebec City corridor, and less often in the Vancouver area. Maximum 1-hour levels of ozone and the number of

Table 6.2.2

Acid Exposures Measured In "Acid Episodes" (Exposure Expressed in Microgram-Hours per cubic Metre of Sulphuric Acid Equivalents)

Site	H^+ concentration ($\mu g/m^3$)	Duration (hours)	C X T Exposure ($\mu g/m^3$)-h
Nova Scotia (Sept. 26, 1983)	9.0	24	216
Dunnville (July 25, 1986)	27.4	12	329
Toronto (July 25, 1986)	19.4	25.3	338
Dunnville (Summer 1988)	16.0	24	382
Pembroke (Summer 1988)	14.0	24	336
Leamington (Summer 1988)	7.4	24	176
Egbert (Summer 1988)	15.5	12	188

hours exceeding 82 ppb are given for some Canadian cities in Table 6.2.3.

More recent ozone measurements in southwestern Ontario indicate that the highest ozone concentrations may occur in the central portion of this region through London, Ontario (Keeler et al., 1989). Ozone measurements at Long Point and Dunnville during the summer months have exceeded 150 ppb for one hour. During the CARES study, it was observed that multiple hours of ozone concentrations greater than 100 ppb were reported several times over the course of the 42 day study. The hourly ozone concentrations were measured continuously during the study. It is also of interest that the ozone maxima typically occurred later in the afternoon (into the early evening) in comparison to other high ozone locations. These late afternoon maxima are indicative of long-range transport of pollutants and precursors and in- transit photochemical

Table 6.2.3

Ozone One Hour Maximas and Hours >82ppb for Selected Urban Sites (1987 or 1988)

City	Max. O_3 (ppb)	Hrs >82ppb	Year
Halifax	122	3	'87
Montreal	118	20	'87
Montreal	121	15	'87
Windsor	159	189	'88
London	137	149	'88
Toronto (North York)	177	157	'88
Oakville	185	122	'88
Winnipeg	80	0	'87
Saskatoon	64	0	'87
Edmonton	92	8	'87
Calagry	84	2	'87
Vancouver	167	34	'88
Vancouver (Pt. Coquitlam)	213	31	'88

From Dann (1989)

transformations. This conclusion was also reached by Anlauf et al. (1985) after analyzing the ozone and other pollutant data from the Nanticoke site, approximately 40 km west of Dunnville. Sites closer to source areas, in which the ozone and other photochemical species peak much earlier in the day, are experiencing more local photochemical production.

Certain areas in southern Canada probably experience appreciable amounts of both local and transported ozone, and would be expected to exhibit two high points each day in their ozone concentration profiles. A very important question in estimating exposures to ambient ozone would be "What critical measure should be used in order to be as predictive as possible of likely health hazard?" The hourly maxima might not be a good indicator of biologically-effective exposure, as the peak hour will occur at different times of day or evening depending upon the geographic relationship between the source or sources and the region in which the exposure is being estimated. Additionally, as we have already discussed, there is now appreciable evidence that the damage produced by ozone in the respiratory system can cumulate over periods of at least 6 hours.

6.2.3 LRTAP HEALTH EFFECTS CONCERNS IN CANADA

The effects of acid particulates and other long range transported air pollutants, as detected in epidemiological studies of children in rural areas of southwestern Ontario, are potentially of concern. The magnitude of the observed difference in both FVC and $FEV_{1.0}$ in the Stern et al. (1989) and continuing 10-city studies--in the range of 1 - 2%--is not large but potentially is of significance as an overall change in a population average, depending on how the change in lung function is interpreted:

(A) The observed lung function differences might reflect a short-term reversible response -- in which case they would primarily be of significance for people, who for other reasons, have compromised lung function. Much higher levels of acids than occur at ambient levels are required, however; to produce such short-term changes in lung function, and these studies were carried out in winter when ambient pollution levels were lowest, lessening the possibility that the observed change was a short-term response.

(B) A more likely possibility is that the difference in lung function reflects a chronic, slowly reversible process. That is, the 2% difference may reflect a relatively long-term response to the continuing input of irritating acid particles which may not increase unless the acid exposures increase. If a 2% difference in $FEV_{1.0}$ in 7-12 year old children were to be carried over without any further increase or decrease into adulthood, it would correspond to the lung function decline produced by about 2 years of normal aging (Dockery et al. 1988).

(C) Another possible interpretation is that the 2% difference among communities might be indicating a continuing progressive change, which might widen as the children got older. Interpreted as a progressive loss per decade of life, the effect would be about a 20%

increase in the age-related decline in lung function -- about the same as would be produced by a pack per day of active cigarette smoking. By further comparison, the effect of passive exposure to maternal smoking in groups of children is to decrease $FEV_{1.0}$ by approximately 0.8% in 6-10 year olds (Berkley et al. 1986; Stern et al. 1989).

In contrast to the situation for acid, short-term responses to ozone clearly are of importance. In several Canadian communities, current peak ambient ozone concentrations are sufficient to elicit measurable transient changes in lung function, respiratory symptoms, and airway inflammation in healthy people engaged in normal outdoor exercise and recreational activities. Furthermore, the effects of ozone on transient functional changes are sometimes greatly potentiated by the presence of other environmental variables.

Although there is a great deal still to be answered about the health effects of LRTAP, there is now converging evidence to support the assertion that LRTAP, in Canada, contributes to the observed effects on people's health ascribed to air pollution. It is biologically plausible to relate the animal toxicology studies to humans, the results of chamber trials on humans indicate that even extremely low levels of ozone can damage tissue in the respiratory system (Kehrl et al. 1989; Utell, 1989), and epidemiological studies show small but measurable decreases in children's lung function in areas where LRTAP levels are elevated.

6.3 INDIRECT HEALTH EFFECTS OF ACID DEPOSITION

Acid deposition may affect human health indirectly through the mobilization of metals from soil, ore bodies and plumbing systems into drinking water, and possibly through the increased bioaccumulation of some metals in food.

6.3.1 TOXIC ELEMENTS MOBILIZED BY ACID DEPOSITION

The indirect exposure of Canadians to the effects of acid deposition could occur through drinking water and ingesting plants, fish, and shellfish containing high levels of heavy metals such as cadmium, mercury, lead, arsenic, chromium, antimony, selenium, copper and zinc. Some of these metals (cadmium, lead, mercury, arsenic and chromium) are considered very toxic (More, 1986; Suess, 1986). Others, like copper, zinc, aluminum, manganese, and iron are presently considered to be detrimental to water potability, rather than a hazard to human health.

6.3.1.1 CADMIUM

Cadmium that has been mobilized by acidic deposition may move into water sources and also food, particularly leafy vegetables and grains (Goyer, 1990; NAPAP, 1990). The major route of exposure to cadmium in general is through food (Friberg et al. 1986). The primary health effect that may be due to exposure to cadmium in food and water is chronic renal disease (nephropathy) which results from the accumulation of cadmium in the cortex of the kidney. Transplacental effects may occur in pregnant women under specific conditions, but the placenta is generally considered a barrier to cadmium. Cadmium accumulates in the body and is only slowly excreted, the result being an increasing body burden as age increases. Increases in the intake of cadmium via food or water would increase the body burden of cadmium.

6.3.1.2 LEAD

Lead is toxic to a number of systems in the body, principally the central nervous system (CNS), the hematopoietic system (blood), and the kidneys (Goyer, 1990; NAPAP, 1990). Lead can have subtle effects on the CNS such as peripheral nerve dysfunction in adults or neurobehavioural and developmental effects in children. In the blood, lead can affect normal synthesis of heme which is necessary for transport of oxygen. Lead can cross the placenta to enter the fetus, and the developing nervous system may be more sensitive to lead than other organ systems (Krigman et al. 1981; Holtzman et al. 1984).

6.3.1.3 MERCURY

Mercury is toxic in its inorganic form but a greater toxic threat to humans is from the organic form, methylmercury. An increase in the acidity of lake water causes an increased conversion of inorganic mercury to methylmercury which can then be accumulated by fish and subsequently biomagnified through the food chain to man (Wood, 1985; Goyer, 1990). A relationship between acid lake water in Sweden and levels of methylmercury in fish suggests that a lower pH may increase the potential of some metals to bioaccumulate (Jernelov, 1986). Thus, the main source for indirect health effects from acid-mobilized mercury may be through the food chain. The major clinical features of methylmercury intoxication are neurologic; paresthesia, ataxia, dysarthria, and deafness. The unborn fetus is more sensitive to methylmercury exposure than adults (Goyer, 1990). In the methylmercury poisonings that occurred in the Minemata Bay region of Japan, infants that were exposed prenatally suffered extensive brain damage while the mother exhibited slight or no symptoms (Harada, 1977).

6.3.1.4 ARSENIC

Arsenic can cause lesions of the skin that are initially non-cancerous but can become malignant (USEPA, 1987). The lesions are associated with excessive exposure to arsenic like that observed from chronic ingestion of water containing arsenic.

6.3.1.5 ALUMINUM

The potentially harmful effects from increased aluminum mobilization by acid precipitation would be from the toxic effects of aluminum on the central nervous system. The pathophysiology of aluminum is not clearly understood nor is its etiology in the diseases for which aluminum has been implicated. These diseases are dialysis dementia, amyotrophic lateral sclerosis, Parkinsonism-dementia syndromes of Guam, and Alzheimer's disease (NAPAP, 1990). The above syndromes are ranked in order of the relative certainty of the role that aluminum may play in the disease. Without a better understanding of the relationship aluminum may play in the pathogenesis of these syndromes it is difficult to speculate on possible health effects from increased aluminum mobilization due to acid deposition.

6.3.1.6 CHROMIUM

Chromium, an abundant element in the earths crust, can cause specific necroses of the proximal convoluted tubules in the kidney (Hook and Hewitt, 1986). Ischemia and tissue damage are observed on the surface of the kidney after exposure to low doses of chromium. As the dose of chromium increases the toxic effects are observed throughout the proximal tubule. Chromium is associated with cancers of the respiratory tract in individuals occupationally exposed to inhaled chromium dusts, but whether chromium causes cancers at other sites is unclear (Goyer, 1986).

6.3.2 BIOAVAILABILITY OF TOXIC METALS

The potential toxicity of a contaminant is more related to its bioavailability than to its absolute concentration in the aqueous environment. Although increased aluminum levels apparently associated with acid deposition are not necessarily a health problem, higher amounts of Al^{3+} in more acid water may induce some toxic effects such as dialysis encephalopathy (Nieboer and Sanford, 1984). Numerous studies (Driscoll, 1980; Johnson et al. 1981; Campbell et al. 1983; Loescher, 1984; Andelman and Miller, 1986; Cole and Taylor, 1986) have all demonstrated accelerated release of aluminum species in freshwater at pH levels below 5.5. Monomeric aluminum has a strong affinity for fluoride ions and dissolved organic acids. The potential toxicity of Al^{3+} may be decreased by its binding to these ligands. Studies such as that of cadmium speciation in river water (Moore and Ramamoorthy, 1984) and lead speciation (Hem, 1976) could be increasingly important to establish actual metal toxicity in areas influenced by acid deposition.

Canadians may also be exposed to toxic heavy metals introduced through acid deposition from sources other than drinking water. As was noted above, methylmercury may accumulate through the food chain and affect people through the food they eat.

One common effect of acidified freshwater is corrosion of distribution systems with resultant solution of heavy metals. This is only a concern for unregulated water supplies, as public water is treated and the pH neutralized before it enters the distribution system. Studies in Ontario on privately supplied lake water showed that water left undisturbed in the distribution system for several days had elevated levels of dissolved copper, lead, and cadmium that often exceeded the recommended safe guidelines (Health and Welfare Canada, 1987; Meranger et al. 1983). Flushing of systems for five minutes after an undisturbed period decreased heavy metal concentrations to below the health guidelines.

Private, essentially untreated drinking water in those areas most influenced by acid deposition can be very corrosive. Havas (1983), has suggested that appreciable metal can be liberated from private distribution systems supplied by wells in Ontario. Static drinking water supplied from shallow wells sampled in spring and fall in southern Nova Scotia had copper and zinc levels above 1 mg/l. Lead, arsenic and mercury were below the recommended health guidelines (Meranger et al. 1986). Although drilled bedrock wells are generally the most hydraulically isolated from the impact of acid deposition they can receive heavy metals, like arsenic, from mineral sources in the aquifer (Meranger et al. 1983; Cherry, 1984). Other inorganic pollutants, such as nitrate, may enter ground water in large amounts through agricultural activities.

6.3.3 RISK TO CANADIANS FROM INDIRECT EFFECTS OF ACID DEPOSITION

Surface water and to a lesser extent ground water appears to exhibit changes which reflect the impact of acid deposition. There has been progressive acidification of lakes over the past thirty years in Canada. At present the most detrimental effect on drinking water may be the corrosion of metal plumbing systems and the release of potentially toxic heavy metals, especially lead, into distributed water.

The areas of Canada most sensitive to acidification include northern Ontario, southwestern Ontario, the north shore of the St. Lawrence River in Quebec, and areas of New Brunswick and Nova Scotia. An estimated 300,000 people in these areas obtain their drinking water from unregulated sources that may be affected by acid deposition. More monitoring of such water supplies for the presence of toxic heavy metals is required before the risk to public health can be evaluated. More studies on metal speciation, bioavailability, and bioaccumulation are needed to determine if acid deposition is causing increased contamination of fish and other foods.

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